Measuring the Therapeutic Supplementation of Ubidecarenone against SOD-1 Mediated Paralysis in ALS C. elegans

Synopsys Science Fair Project #: 091-680-52

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Using worms with ALS, we tested whether Coenzyme Q10 could improve their survival and wriggling movement, since ALS is a motor disease. We found Improvement with high amounts of Coenzyme Q10.

Amyotrophic Lateral Sclerosis, or ALS, is a neurodegenerative disease that affects roughly 20,000 people in the US alone each year; yet despite its ubiquity, no effect treatments exist at all on the market to reverse paralyzing progression in the disease. Our project is aimed at finding a potential treatment to ALS within common dietary supplement coenzyme Q10, a factor in the electron transport chain that has long been theorized and consumed for its benefits in reducing many cardiovascular diseases. We theorize that CoQ10 supplementations on model organism C. elegans with an ALS gene mutation will ameliorate indicated paralysis in the roundworm.

In the first few weeks, C. elegans were chunked from a stock plate and then cultured in separate NGM plates previously seeded with agar and a lawn of OP50 E. coli (nonpathogenic) in LB. From there, serial dilutions were used to create four varying concentrations of diluted CoQ10 (fat-soluble) with extra virgin olive oil solution: 0mg./mL., 0.1mg./mL., 0.2mg./mL., and 0.4mg./mL. On a glass slide under a microscope, C. elegans were then isolated by identification of age –fat L4 stage C. elegans (adult) demonstrating quintessential movement in the plate– and counted for bends over a minute, with bends as a behavioral assay for locomotion and paralysis in the worms. Wild-type C. elegans (no ALS) without treatment were quantified eight separate times over a week and averaged for bends and compared to the average of eight trials of bends for the ALS-gene (SOD-1) C. elegans without any treatment; the results showed a significant average decrease in the amount of bends for SOD-1 C. elegans, indicating a presence of the ALS-mediating gene mutation. A subsequent T-test and SEM analysis in Prism 6 was made to verify these results; however, the p-value was more than 0.005 and the relationship was deemed insignificant and unsupportive of the presence of ALS in SOD-1 C. elegans, likely due to a high variation in SEM for the Wild-type group, but not ruling out the validity of the SOD-1 Control group.

Next, SOD-1 C. elegans were treated with varying concentrations of Coenzyme Q10 solution as well as a fluorescein sodium solution used as a UV marker to confirm the consumption of CoQ10 (later mixed onto the same plate with CoQ10 solution). Each of these concentrations were treated to one specific plate of C. elegans, and from there bends were quantified. After one week of treatment, we found C. elegans treated with any amount of CoQ10, on average, demonstrated significant more locomotion than that of the control group. Our results with checked with Prism 6 ANOVA multiple-comparison T-test. Results indicated significance between several groups, most notably between some groups of increasing concentration and between the group SOD-1 Control and SOD-1 0.4mg/ml. This demonstrates a verified amelioration in ALS-mediated paralysis in concentration 0.4mg/ml, supporting our hypothesis –at least in the greatest concentration– that CoQ10 does have an effect on reducing ALS.